ETIOLOGICAL FACTORS IN CEREBRAL PALSY*

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CEREBRAL palsy, by definition, indicates a condition caused by pathology to the motor control of the brain. The neuromuscular manifestations may be spastic or flaccid paralysis, weakness, incoordination or ataxia.

HISTORY

The story of those having cerebral palsy is one of children being hidden away in the home as a curse on the family or sent to mental institutions. Medieval mosaics, monuments and paintings give evidence that cerebral palsy was known many long years ago.

It was not until 1862 that William John Little, an English surgeon wrote a monograph describing the condition which we now call cerebral palsy. The spastic grimacing, drooling child with a "scissors gait" that Dr. Little described gave the erroneous impression that all children with cerebral palsy were of that type and feebleminded. The term Little's disease should not be used to describe all children with cerebral palsy since we know that impaired intelligence does not necessarily accompany the motor disability and that spasticity is only present in one type of cerebral palsy.

INCIDENCE

It is difficult to give the number of children having cerebral palsy. Surveys have always revealed that the number in any given area is always greater than the number reported; that the incidence is not affected by economic, social or geographical factors, and since the condition does not occur in epidemics the number of cases occurring is approximately the same from year to year.

On the basis of repeated samplings, Dr. Phelps estimates that at a

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birth rate of twenty-three per thousand live births, seven cases of cerebral palsy are born each year in each 100,000 of the total population. One of these children dies before the age of six. Using this formula, it is estimated there are 104,895 cerebral palsied persons in New York State up to thirty-five years of age.

The survey made by the New York State Health Department verified Dr. Phelps' estimates that one infant in every two hundred births shows the clinical signs of cerebral palsy.

Etiology

There are certain predisposing factors related to cerebral palsy. It occurs more frequently in premature children, the first born and those with heavy birth weights where prolonged labor is more frequent. It likewise occurs more often in infants of older women, and in boys more frequently than in girls. Older women, on the average, have heavier babies, and boys, on the average, weigh more than girls at birth. Cerebral palsy seems to occur more frequently among white children than colored and this may be due to the average smaller size of the colored new-born and the lower incidence of the Rh negativity in the colored race. With these exceptions there seem to be no economic, social or geographic predilections for cerebral palsy.

The *Prenatal* causes may be due to hereditary or congenital factors. The *hereditary* causes of cerebral palsy may be due to anomaly of the germ plasm resulting in abnormal development of the fetal brain. The *congenital* or *nonhereditary* factors which may cause cerebral palsy are as follows:

1. *Anoxia.* Anything that interferes with placental circulation is a potential cause of anoxia. The brain, primarily the basal nuclei, and their connections, are highly sensitive to the lack of oxygen; compression of the cord between the head and bony pelvis or kinks in the cord, could prevent adequate circulation to the fetal brain. Threatened abortion, bleeding during pregnancy or premature separation of the placenta is frequently found in mothers giving birth to cerebral palsied infants.

2. *Maternal Infectious Disease.* Any maternal infection, especially with a neurotrophic virus, may involve the fetal brain. German measles during the first three months attacks the brain of the fetus with great regularity.

3. *Maternal Metabolic Disease.* One of the most frequent causes of prenatal intracerebral hemorrhage is toxemia of pregnancy. The fetal blood vessels in the brain are affected by substances in the mother's blood vessels with death of brain tissue and secondary hemorrhage. Toxemia of pregnancy predisposes to prematurity. Maternal diabetes may cause injury to the fetal brain. Diabetic mothers usually have infants with excessive birth weights and more frequently develop toxemias of pregnancy.

4. *Erythroblastosis Fetalis.* Rh refers to an additional blood factor which is present in 85 per cent of the white population. When an Rh negative woman bears an Rh positive child, the mother often develops anti-Rh positive antibodies which cause a typical form of cerebral palsy. Only one of approximately thirty-three children born to Rh negative women develops erythroblastosis fetalis. The reason for this small number is that the first born is not affected, the first born are more plentiful than the second born and a certain number of the children born will themselves be Rh negative.

The result of Rh incompatibility of the parents often produces kernicterus or neonatal nuclear jaundice of the brain. As a result of the anemia and damage to the liver there is interference with the nutrition of the brain, usually the basal nuclei. When this area is damaged the athetoid type of cerebral palsy occurs often associated with deafness and paralysis of the eye muscles.

The *Natal Causes* of cerebral palsy may be due to 1) anoxia; 2) blockage of the respiratory passage causing asphyxia; 3) analgesia; 4) trauma due to cephalopelvic disproportion and delivery of the breech before the head and forceps; 5) sudden changes in pressure; 6) prematurity and 7) low vitamin K level.

1. Anoxia is the most important natal factor. When the placenta separates from the uterus or the umbilical cord is clamped off the child is dependent upon its own lungs. Kinking or pinching of the umbilical cord during the birth process may result in interference with fetal blood supply and thus cause anoxia.

2. *Blockage* of the respiratory passages causes asphyxia. The newborn child may aspirate a great deal of mucous or amniotic fluid in its passage through the birth canal. Such respiratory obstruction must be recognized and removed by suction or by the passage of a tracheal catheter.

3. *Analgesia.* The respiratory center of the infant is much more sensitive to morphine than the adults'. Many mothers who have been given "twilight sleep" have their child born with morphine poisoning. The child breathes very slowly and the pulse is very slow and the temperature low. Because of insufficient oxygen it will be a "blue baby." Obstetrical anesthesia which causes maternal asphyxia may also asphyxiate the fetus. Spinal and caudal anesthesia may lower the systolic blood pressure below 80 in 10 per cent of cases which is sufficient to reduce seriously the oxygen supply to the baby.

4. *Trauma.* Most of the traumatic causes of brain injury at birth may be considered as physiologic. Just being born is a difficult hurdle to pass. In the birth process, the baby uses its head for a battering ram propelled by strong uterine contractions. When the child's head is large and the pelvis small, the natural safeguards which allow the skull to conform to the shape of the birth canal may be insufficient to protect the brain from injury. This is especially true if labor is prolonged and hard. Occasionally too strenuous an application of forceps by the obstetrician, may injure the skull and brain. Contrary to popular opinion, trauma caused by the obstetrician is probably responsible for less than 5 per cent of all cases of cerebral palsy.

In normal cephalic birth the infant can breathe before the blood supply from the placenta is interrupted. When the breech is delivered first, the time between the delivery of the cord and the delivery of the head becomes a potential period of anoxia. Thus, cerebral palsy is much more common in infants born by breech than by cephalic mechanism.

5. *Sudden Changes in Pressure.* The so-called "spontaneous deliveries" and infants delivered by cesarean section which prove satisfactory to the mother, are not free of complications associated with brain lesions. The intrauterine pressure is greater than atmospheric pressure. A sudden change from the former to the latter may result in sudden release of pressure with resultant air emboli, ruptured blood vessels and hemorrhage. In both instances there may be insufficient time to allow for natural decompression.

6. *Prematurity.* A premature baby has much thinner blood vessels than the full term mature child. The weak blood vessel walls will tend to predispose this infant to cerebral hemorrhage.

7. *Vitamin K.* Sometimes a child is born with a tendency to bleed. This neonatal bleeding may be due to a lack of vitamin K in the blood.

By giving vitamin K to the mother while she is in labor, she in turn will transfer it to the child. This condition used to be responsible for about one per cent of the cases of cerebral palsy.

The *Postnatal* causes of cerebral palsy result from brain pathology after birth. The principal causes are trauma, infections, neoplasms, drugs, vascular conditions and anoxia. It is estimated that there are over one million patients with acquired cerebral palsy of the spastic and flaccid hemiplegia type as a result of cardiovascular accidents. This number increases with our life expectancy.

Discussion

Cerebral palsy is difficult to diagnose in the infant and very young child. By obtaining a careful family history in our cerebral palsy clinics for children, efforts are made to discover the indications for suspecting brain damage.

The developmental history of the child often indicates brain pathology. Gessel and Amatruda have listed the periods in which infants perform certain movements. The failure of the child to follow the developmental maturity scale is often the only early indication of brain damage. If a baby cannot lift his head upwards from the prone position for short periods during the first weeks, kick both feet together instead of a reciprocal pattern, or holds his hands in a closed position after sixteen weeks, or is delayed in sitting, standing and walking beyond the normal periods, it must be regarded as a significant sign of central nervous system impairment.

Conclusions

The number of infants born with cerebral palsy, which is one in every two hundred live births, can only be reduced in so far as we are able to control the predisposing factors, and the prenatal and natal causes.

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